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Title

Understanding the influence of neurodevelopmental disorders on offending: utilizing developmental psychopathology in biosocial criminology

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Abstract

Evidence from a wide range of countries consistently suggests a disproportionately high prevalence of neurodevelopmental disorders amongst young offenders in custodial institutions. This indicates an increased rate of serious and persistent offending amongst young people with diagnosable disorders, and therefore a failure of current policies and practices to address this vulnerability. Given this high prevalence it is vital to seek better understandings of the trajectories of offending experienced by young people with specific disorders. Biosocial criminology is uniquely placed to examine this relationship, given its emphasis on the influence of biological processes on antisocial behaviour and the role of social and environmental contexts in shaping the course of these processes. However there are significant challenges and limitations to effectively modelling the complexity and heterogeneity of the influence of neurodevelopmental disorders using dominant biosocial methodologies. Addressing these concerns necessitates improved understandings of the etiology, expression and progression of particular neurodevelopmental disorders, as evident in developmental psychopathology. Understanding the particular combination of biological processes apparent in the progression of specific disorders and their influence on specific components of social functioning can inform more effective biosocial models of criminal behaviour in the context of neurodevelopmental impairment.

Keywords: neurodevelopmental disorders; neurocognitive impairment; neuropsychological impairment; developmental psychopathology; biosocial criminology.

Childhood neuropsychological impairment can occur when there is a compromise of the central nervous system – which consists of the brain, the spinal cord and a related set of neurons - or peripheral nervous system - which sends sensory information to the brain and controls the functioning of organs and muscles (Patel, Greydanus, Omar, & Merrick, 2011). Such compromises are often the result of a complex mix of influences, including genetic, pre-birth or birth trauma, illness or injury in childhood, or nutritional, educational or emotional deprivation, and might result in one or more of a wide range of physical, mental or sensory functional difficulties. Common symptoms include: cognitive deficits; specific learning difficulties; communication difficulties; and emotional and behavioural problems (Patel, et al, 2011; American Psychiatric Association [APA], 2013). This broad range of impairments incorporates an array of clinically defined disorders or conditions, including (though not restricted to): intellectual disability; specific learning difficulties; communication disorders; attention-deficit/hyperactivity disorder (ADHD); autistic spectrum disorder; traumatic brain injury (TBI); and foetal alcohol syndrome disorders (Patel, et al, 2011; APA, 2013).

A recent comprehensive review of evidence across a range of international contexts reveals consistently high incidence rates of childhood neurodevelopmental disorders amongst offending populations (Hughes, Williams, Chitsabesan, Davies, & Mounce, 2012). Following an extensive review of research from a variety of academic disciplines, as well as evidence published by health and justice organisations and government departments, Hughes et al (2012) compare the rates of specific neurodevelopmental disorders amongst young people in custodial institutions to those within the general youth population. In doing so, they demonstrate a disproportionate prevalence of a range of disorders amongst young people in custody. The findings of this review are summarized in Table 1, alongside a basic definition of each disorder.

INSERT TABLE 1 HERE

This data clearly requires careful interpretation given the methodological and analytical challenges in combining and comparing studies with varied definitions, measures, methods, populations and national cultural and policy contexts (Hughes et al, 2012; Fazel, Doll, & Langstrom, 2008). In addition, utilising clinical definitions of neurodevelopmental disorders may mask a broader range of impairments. The Diagnostic and Statistical Manual of Mental Disorders (DSM; APA, 2013) provides the most commonly utilised classification system for such disorders. However, the precise clinical definition of specific disorders can contrast to the fluid and overlapping nature of the symptoms of impairment experienced by individuals (Kruger, Watson & Barlow, 2005), or make invisible those young people with 'subclinical' levels of needs, yet very real difficulties. In part this is addressed by the shift in the most recent DSM to a dimensional definition that reflects variation in specific symptoms (Reigier, 2007), but such an approach is not typically reflected in research to date.

Notwithstanding these challenges, the weight of evidence is such that it is imperative to seek explanations as to why young people with certain neurodevelopmental disorders are at such increased risk of custodial intervention, and in doing so to better explain the trajectories into serious and persistent offending experienced by young people with neurocognitive and neuropsychological impairments. This necessitates an interface between criminological theories concerned with the dynamic and cumulative influence of risk and protective factors on offending pathways, and the understandings of the etiology, expression and progression of particular neurodevelopmental disorders evident in developmental psychopathology. The following discussion will argue that biosocial criminology is uniquely placed to integrate

these understandings, given its emphasis on the influence of biological processes on antisocial behaviour, and the role of social and environmental contexts in shaping the course of these processes. However, limitations and challenges within current approaches suggest value in applying developmental psychopathology, so as to understand the progression and expression of specific disorders and therefore inform biosocial models better able to explain trajectories of offending for young people with neurocognitive and neuropsychological impairments.

Such an application builds on a long history of interdisciplinary approaches in criminology, including a number of integrated general theories of crime that draw on advances across the social sciences, psychology and biology (see, for example, Henry & Lukas, 2009 and Robinson & Beaver, 2009). It is not the intention of this paper to replicate such general theories, but rather to supplement them by arguing for the specific value in utilizing advances in developmental psychopathology – which are typically absent in integrated general theories of crime - to explain criminality among those with atypical neurodevelopment.

Utilizing biosocial criminology to examine neurodevelopmental impairment

Understandings of the complex influences of neurodevelopmental impairments on offending behaviour are inadequately addressed by the dominant, traditional theories of criminology. In part this reflects a lack of focus on biological explanations for criminality. Wright and Boisvert (2012, p.1235) argue that ‘despite tremendous evidence to the contrary’, prevailing criminological approaches ‘simply overlook or ignore the individual characteristics that differentiate offenders from nonoffenders.’ Instead, ‘Environmental theories that largely ignore individual differences (especially differences linked to biology) dominate mainstream

criminology' (Walsh & Ellis, 2007), leading to a concentration on 'experiential factors' that, whilst not inevitably excluding the biological, 'do not provide a model that would accommodate their consideration.' (Fishbein, 2006, p.48) Furthermore, even those theories that emphasise sociological and experiential explanations are critiqued for failing to consider experiences related to impairment and disability (Dowse, Baldry & Snoyman, 2009).

Biosocial criminology is in a unique position to address this deficit in understanding of impairment-related influences on offending. Biosocial criminology seeks to 'understand how biological processes matter in the etiology of antisocial and criminal behavior, how these processes shape and are shaped by environmental features, and how individuals develop over the life-course.' (Wright & Cullen, 2012, p.245) Key to this is an understanding of ontogeny: 'the origins and life-course development of an individual organism' (Wright & Cullen, 2012, p.246). The study of ontogeny suggests that 'much human development is preprogramed and emerges in a somewhat orderly and predictive fashion, sometimes with only limited environmental input.' (Wright & Cullen, 2012, p.246) However, young people engaged in serious and persistent criminal behaviour are seen to 'depart significantly from normative behavioral trajectories' (Wright & Boisvert, 2009, p.1236).

The study of biological influences on offending is therefore the search for explanations of ontogenic diversity that might explain variation in key behavioural traits. An understanding of 'normative development' enables a focus on difference from the 'pre-programmed' norm; that is, on neurological diversity and disadvantage: 'Delays in normative developmental sequences provide researchers with opportunities to better understand how developmental dysfunction is linked to the onset of problem behaviors' (Wright & Cullen, 2012, p.246). Such 'developmental dysfunction' is expressed through 'behavioral, cognitive and

psychological traits, such as impulsivity, attention deficits, aggressiveness, and heightened sensitivity to rewards and stimulation.' (Fishbein, 2006, p.45)

Whilst emphasizing the role of biology in increasing propensity to offend, biosocial models also highlight the importance of social and environmental contexts. The 'immediate social environment' is presented as key to the process of 'human development' which 'occurs in interaction with others across varying social contexts', as our 'traits, behaviors, and propensities emerge and interact with the behaviors and personalities of others' (Wright & Cullen, 2012, p.246). Biosocial models consistently demonstrate that 'the presence of both [social and biological] risk factors exponentially increases the rates of antisocial and violent behavior.' (Raine, 2002, p.311) Whilst biological risk factors may be present within a large population, their impact might therefore be more realized in people in adverse environmental conditions (Fishbein, 1996). 'In other words, individuals with genetic disadvantages or risk traits may be more violent or antisocial in a "criminogenic" environment than others under similar circumstances.' (Fishbein, 1996, p.92) The opposite causal pathway is also apparent: 'environmental exposure to certain risk factors is not always random but is instead produced by individual genetic propensities' (Wright & Cullen, 2012, p.246). For example, Caspi et al (2002) found that high levels of monoamine oxidase A (an enzyme that metabolises neurotransmitters including dopamine and serotonin) moderate the effect of maltreatment in childhood on future antisocial behaviour. Thus:

> 'Biosocial studies help to better specify not only which environmental risk factors are important, but also why specific children, for example, are harmed by specific environmental risk factors although other children, exposed to the same risk factors, remain resilient.' (Wright & Cullen, 2012, p.247)

The relevance of biosocial criminology to the study of neurological impairments is therefore clear. Neurodevelopmental disorders are indicative of atypical ontogeny and, as illustrated in table 1, commonly relate to a range of 'developmental dysfunctions'. Many of the biological factors that have been the focus of fruitful research within biosocial criminology are of direct relevance to the etiology, symptoms and expression of specific neurodevelopmental disorders, including brain function and structure (e.g. Crowe & Blair, 2008, Wilson & Scarpa, 2012) and executive functioning (e.g. Ganesalingam, Sanson, Anderson, & Yeates, 2007, Meltzer, 2007). Furthermore studies have illustrated the interaction of biological and social and environmental factors in heightening risk of offending in the context of specific disorders, such as the influence of parent-child interactions following TBI (e.g. Wade et al, 2011, Yeates et al, 2010).

The challenges and limitations of current biosocial criminology

This unique focus on a wide range of potential biological explanations for antisocial and aggressive behaviour therefore ensures biosocial criminology is well positioned to contribute towards enhanced understanding of the apparent heightened risk of criminality experienced by young people with neurodevelopmental disorders. Indeed there are examples of biosocial criminological research that strongly emphasise neuropsychological impairments in examining offending. This is most apparent in the work of Terrie Moffitt and colleagues in considering the 'dual taxonomy' of 'adolescence-limited' (AL) and 'life course persistent' (LCP) offenders (Moffitt, 1993, 2006; Raine et al, 2005). Moffitt (1993, p.681) uses the term 'neuropsychological' to 'refer broadly to the extent to which anatomical structures and physiological processes within the nervous system influence psychological characteristics

such as temperament, behavioral development, cognitive abilities, or all three.' 'Deficits' in these psychological characteristics are found to be 'linked to the kind of antisocial behaviour that begins in childhood and is sustained for lengthy periods' (Moffitt, 1993, p.680), yet largely absent amongst those whose offending begins and ends in adolescence.

Whilst illustrative of the potential for biosocial criminology to contribute to this research agenda, consideration to this body of research also reveals some of the significant challenges and current limitations in utilizing dominant biosocial methodologies in order to understand the influence of specific neurodevelopmental disorders on offending behaviour. As detailed below, these challenges and limitations relate to a reductionist conceptualization of impairment that is unable to provide a holistic or heterogenic representation of specific disorder, and typically presents impairment as fixed or static over time and consistent across various social contexts.

Whilst drawing attention to a range of highly relevant biological factors, the methodologies popularly utilised in biosocial criminology do not readily support a holistic consideration of the myriad of biological processes implicated in neurodevelopmental disorders. Such research seeks to identify 'factors in a child's life that, within large population samples, have a statistical correlation with anti-social or offending behaviours' (Prior & Paris, 2004, p.15). In seeking to identify such factors, criminological studies typically focus on specific aspects of biology, such as genetics, neurotransmitters or specific cognitive skills. The reduction of such neurological and biological functions and processes to measurable indicators also overly simplifies the complexity of their role. For example, Syngelaki et al (2009, p.1214) argue that there has been insufficient attention to 'the role of specific frontal subregions' or 'different forms of executive function' in studying 'aggressive and antisocial behaviour'. This is a

consequence of the complexity of the multitude of potential explanations for criminality, the need to test specific hypotheses, and the finite potential to capture relevant indicators. Nonetheless it does not support a ready account of the complex interactions between various components of biological systems, or how these interactions result in particular dysfunctions and behaviours observed in the symptoms and expressions of specific neurodevelopmental disorders.

Such a critique can be applied to the dual taxonomy of LCP and AL offenders. Moffitt's binary classification has been widely applied and highly influential. A large number of empirical studies have reaffirmed the existence of populations of offenders with such trajectories, with reviews of such studies suggesting the supporting evidence for the 'dual taxonomy' to be strong (Moffitt, 2006). However, the classification has also been the subject of various critiques and attempts at reconceptualisation (see Skarðhamar, 2009 for a review). In particular the ability of this dichotomous categorisation to effectively represent a typology of offenders or criminal careers has been questioned, with the 'discrete phenomena' of AL and LCP offending seen to be at odds with the complexity of the various variables presented as explaining the distinction (Skarðhamar, 2009, p.8).

This general critique is pertinent to the particular concerns of this paper. The simple dichotomous categorisation of offenders fails to account for the continuum of expression in the three types of neuropsychological characteristics that are considered, and their myriad of component 'sub characteristics', or to represent the multidimensional nature of these complex constructs. Whilst this is to be expected of a more general theory of offending, concerned with a broader range of factors and explanations, it poses several challenges in making sense of the particular influence of neuropsychology. It is unclear whether the model assumes a

particular, singular cut-off point at which a young person is seen to have a neuropsychological 'deficit' and, if so, how this might be defined in relation to each characteristic; 'no such thresholds are discussed nor specified by Moffitt' (Skarðhamar, 2009, p.8). The precise relationship between neuropsychological 'deficit' and LCP offending is therefore unclear. Of course, deficits in these characteristics are evident in the expression of various neurodevelopmental disorders. If such a threshold does exist, it might be surmised that all those with specific, clinically defined neurodevelopmental disorders would be considered to have such neuropsychological deficits, yet this is to simplify the heterogeneity of such disorders and their influence on behaviour.

The apparent assumption of homogeneity is also reflected in a restricted understanding of the etiology of identified neuropsychological deficits. Whilst recognising the role of 'anatomical structures and physiological processes' (Moffitt, 1993) in determining such deficits, these factors are not considered, being beyond the scope of the methods used. The role of social and environmental factors in influencing neuropsychology are also noted, though inadequately modelled. For example, Raine et al (2005) search for associations with 'important environmental processes' such as childhood 'abuse' or 'psychosocial adversity', history of head injury', though, unsurprisingly, simple links between these variables and impairments are not found. By building upon biosocial criminological approaches that have successfully modelled the interaction of biological and social factors in heightening risk of offending, there is the potential to more accurately model associations between biological attributes related to specific neurodevelopmental disorders and social and environmental risks.

Theorisation of the role of neuropsychological characteristics in the long-term trajectories of offenders is further simplified by the assumption that such deficits are present in childhood, prior to the early onset of offending. Whilst empirical evidence suggests this to be the case for identifiable populations of LCP offenders, such a theorisation is unable to account for neurodevelopmental difficulties that become apparent later in childhood, either as a result of injury or illness, such as in the case of TBI, or because they are cumulative in nature, such as specific learning difficulties or particular communication disorders. The role of the late onset or cumulative progression of neuropsychological characteristics on adolescent offending is not accounted for in a model that assumes LCP offending begins in childhood. It is unclear whether an adolescent experiencing a later onset of neuropsychological deficits is at greater risk of life course persistent offending, or whether, if engaging in criminality, this is still likely to be adolescent-limited, as suggested by the simplified classification. More importantly, explanations for these patterns are required, including consideration to the mechanisms that might protect young people with late onset neuropsychological deficits from persistent adult offending.

The inadequacy of the 'dual taxonomy' to account for all observable trajectories in offending amongst offenders with early childhood neuropsychological deficits that pre-exist antisocial behaviour is recognised in more recent work by Moffitt and colleagues. For example, Moffitt's (2006) review of research suggests the common identification of an additional category of 'childhood limited' antisocial individuals who demonstrate neurocognitive impairments but refrain from offending in adolescence. This provides further evidence of the complexity of expression and progression of neurodevelopmental difficulties and therefore the need for a more nuanced examination of the role of neuropsychological deficits in trajectories of offending. As Raine and colleagues (2005) argue, particular attention is needed

to the resilience to offending amongst this group, including to any social or environmental protective factors supporting desistence despite neuropsychological deficits.

Whilst the concept of 'life course persistency' necessitates a longitudinal approach that implies a developmental perspective, research examining this hypothesis has presented neuropsychological deficit as a fixed and static category. This is evident in the work of Raine and colleagues (2005) in examining 'Neurocognitive Impairments in Boys on the Life Course Persistent Antisocial Path'. In this study, neurocognitive impairments are measured at age 16-17 despite the model suggesting that it is the existence of such impairments in childhood that determines the likelihood of LCP offending. Whilst recognised by the authors as a limitation and necessary assumption of the study design, the implication that an impairment in adolescence was present prior to offending demonstrates a lack of consideration to the developmental nature of neuropsychological impairments in such research.

Where developmental approaches have been applied to the study of offending behaviour (broadly in keeping with developmental psychopathology, as described below), such approaches do not typically or effectively utilise such models to understand the complex influence of neurodevelopmental disorders on such behaviour. Most notably, Raine (1993) has applied such a framework directly to the study of crime, with the title of his influential book presenting 'Criminal Behavior as a Clinical Disorder' in itself. This builds upon the identification of the LCP category of offenders by presenting such behaviour as 'psychiatrically disordered' (Howard, Williams, Vaughn, & Edmond, 2004, p.442). However such an approach is broadly critiqued as representing a '(bio)medicalization' of criminality that that consequently ignores 'the socially constructed nature of crime' (Walby & Carrier, 2010). As such, this places criminality as the central focus of attention when, for those with a

neurodevelopmental disorder, it might more accurately be considered as a secondary outcome of behavioural traits and social experiences more directly associated with the disorder itself, as is discussed below.

A developmental framework has also been applied to the study of conduct disorder: a behavioural disorder developed, at least in part, from the identification of a LCP offending categorisation (Bierman & Sasser, 2014; Fairchild, van Goozen, Calder, & Goodyer, 2013). In DSM-V, conduct disorder is defined by a persistent pattern of behaviour 'that violates either the rights of others or major societal norms', including observations of at least three of the following symptoms: 'aggression toward people and animals'; 'destruction of property'; 'deceitfulness, lying, and theft'; and 'serious violations of rules' (APA, 2013). This body of research demonstrates the potential application of developmental psychopathology to antisocial behavioural traits. However, for the context of this article, whilst there is some comorbidity of conduct disorder and specific neurodevelopmental disorders (Bierman & Sasser, 2014; Pardini & Frick, 2013), these conditions are distinct. Consideration to conduct disorder alone is insufficient to examine the role of specific neurodevelopmental disorders in influencing trajectories towards serious and persistent offending.

Representation of neuropsychological impairments as fixed and static over time is mirrored by assumptions of consistency in the expression of the characteristics of these impairments in various social contexts. For example, studies typically assess behavioural characteristics in terms of particular tendencies or dominant traits, assessed through validated scales. This is a necessary and indeed valuable approach. However, Yeates et al (2007, p.537) argue that the presentation of these 'behavioral tendencies' is not consistent, and is instead determined by the context of the social situation and the 'nature of the children's relationships with the

individuals with whom they interact'. For example, a young person will interact differently with friends than with unfamiliar peers, or when 'attempting to enter a peer group activity' as opposed to 'responding to peer provocation' (Yeates et al, 2007, p.538). This leads Yeates et al (2007, p.537) to argue that: 'A detailed understanding of children's social interactions cannot be attained using conventional rating scales or questionnaires but instead requires direct observation in a variety of contexts.' This is particularly the case in the presence of disorders such as autism, ADHD or communication disorders, which affect behaviour in certain types of social interactions. It also has particular and necessary application to an understanding of the contexts in which crime is committed, particularly where influenced by impulsivity, reactive aggression or peer group interactions, as discussed further below. This is, of course, well understood in biosocial criminology. As already discussed, individuals with particular biological disadvantages have been found to be at greater risk of violent or antisocial in a criminogenic environment than others under similar circumstances (Fishbein, 1996). As will be explained, neurodevelopmental impairment seems likely to serve as such a disadvantage.

Whilst clearly not singularly representative of the rich variety of approaches within the discipline, the influential work of Moffitt and colleagues regarding the AL and LCP dual taxonomy therefore provides an illustrative example of the current limitations in the modelling of neurodevelopmental disorders in biosocial criminology. In particular, the heterogenity in expression and progression of neurodevelopmental disorders and the complex relationships between biological, and social and environmental factors in influencing this needs greater consideration in any attempt to model the influence of neuropsychological impairment on criminal or antisocial behaviour. The following discussion will illustrate how

this can be achieved through consideration to emerging understandings of the developmental psychopathology of specific neurodevelopmental disorders.

Developmental psychopathology of neurodevelopmental disorders

Developmental psychopathology is the study of psychological disorders, utilising a life-course framework in order to understand the biological and environmental factors and processes that can cause divergence from normative or typical development and map causal pathways from initial manifestation to full expression of a disorder (Cicchetti, 1993; Rutter & Scroufe, 2000). Informed by 'the emerging field of social cognitive neuroscience', such an approach is seen to supply the 'tools needed to better understand the neural substrates and social-cognitive processes associated with social functioning', whilst also providing 'a foundation for a multilevel, integrative analysis of the social difficulties arising from neurological insults' (Yeates et al, 2007, p.535).

> 'In short, we now have the tools and models to begin to understand how children's daily functioning in the social world is associated with their abilities to identify, think about, produce, and regulate emotions; to consider other people's perspectives, beliefs, and intentions; and to solve interpersonal problems. Furthermore, we can model this association in terms of developmental processes and brain pathology.' (Yeates et al, 2007, p.536)

Developmental psychopathology is therefore necessarily 'an integrative discipline', utilising multilevel and multivariate designs to consider 'the degree of convergence or divergence in

the organization of biological, psychological, and social-contextual systems as they relate to symptom manifestation and disorder' (Cicchetti & Rogosch, 2002, p.7).

Whilst primarily concerned with pathological or atypical outcomes, this also necessitates comparison to normative adaptation and development. To this end, Yeates et al (2007; p.537) propose a general 'multilevel, integrative, heuristic model' of 'the individual characteristics and social skills, interactions, and various aspects of social adjustment that constitute social competence', through which the specific impact of a broad range of 'childhood brain disorders' can be considered, including 'acquired brain injuries' and 'neurodevelopmental disorders'[1]. Whilst variably defined, 'social competence' is broadly conceptualized as 'the ability to achieve personal goals in social interaction while simultaneously maintaining positive relationships with others over time and across situations', while 'social adjustment represents the capacity of individuals to adapt to the demands of their social environment' (Anderson & Beauchamp, 2012, p.4). As such, social competence must be understood as a product of the 'personal characteristics of the child' (including emotional regulation, problem-solving skills, and understanding of the consequences of particular actions), and their engagement with their social world; that is, the 'interactions between the child and members of his or her social world, and the interpretations of the self and others that the child's actions are acceptable and successful.' (Yeates et al, 2007, p.36)

INSERT FIGURE 1 AROUND HERE

[1] The model developed by Yeates et al (2007) serves only as an illustrative example. Other similar models exist – see, for example, Anderson and Beauchamp (2012).

As presented in Figure 1, the model seeks to ‘specify the relations between social adjustment, peer interactions and relationships, social problem solving and communication, social-affective and cognitive-executive processes, and their brain substrates’ (Yeates et al, 2007, p.536). It does so by detailing the key dimensions of three ‘levels’ or ‘domains’ associated with social competence, and the relations between these levels. For example, social information processing is understood to involve ‘distinct problem-solving steps’ employed in response to specific social situations, which necessitate the use of cognitive and affective functions, including executive functions, social communication skills and emotional regulation (Yeates et al, 2007, p.537), while social interaction is seen to be determined by three ‘broad behavioral tendencies’: ‘prosocial, affiliative behavior’; ‘aggressive or agonistic behavior’; and ‘socially withdrawn behavior’ (Yeates et al, 2007, p.538). In considering the ‘developmental trajectories that occur within these domains’ (Yeates et al, 2007, p.536), the model also makes it clear that these characteristics are not fixed or static, but vary over time, as well as in different contexts and social situations.

Having established a general model of social competence through these three ‘levels’ or dimensions applicable to all young people, the model then recognises the role of ‘a variety of risk and resilience factors that can hamper or promote social development’ (Yeates et al, 2007, p.539). It is in this consideration that the particular influence of ‘insult related’ factors on the various aspects of social competence is considered. A particular ‘neurological dysfunction’ is ‘conceptualised as risk factors that increase the likelihood of deficits in social information processing, atypical social interaction, and poor social adjustment’ (Yeates et al, 2007, p.539). Thus the specific influence of particular symptoms and expressions of neurological dysfunctions, as identified in research from various disciplines, can be mapped onto components of the general model so as to explain deficits in social competence or

atypical social adaptation. In parallel to intrinsic, 'insult related' factors, the model also draws attention to social and environmental factors impacting upon social development. Such factors include 'socioeconomic status, parenting behaviours, and parent-child relationships' (Yeates et al, 2007, p.539).

Applying developmental psychopathology to the study of crime

Models of developmental psychopathology such as that of Yeates et al (2007) can be applied to the study of criminal or antisocial behaviour. Understandings of social functioning and adaptation can be utilised to understand criminal or antisocial behaviour as an inappropriate response to a social situation that is indicative of a deficit in social competence. Heuristic frameworks regarding the constituent aspects of social competence can then be utilised in order to identify and understand this deficit as a product of problematic social information processing, social interaction and / or social adjustment. In turn, research examining particular neurodevelopmental disorders can be utilised to identify specific deficits in particular components of the model with an association to criminal or antisocial behaviour that might be explained by 'insult related' factors. This identification supports attempts to theorise the role of neurocognitive or neuropsychological impairment in modelling an explanatory pathway towards offending behaviour and is in keeping with the integrative approach of biosocial criminology, as presented above (Wright & Cullen, 2012; Fishbein, 1996). The following examples illustrate the potential of such research to identify a range of such deficits, including in relation to executive functioning, social-affective functioning, and susceptibility to peer group pressure in certain social interactions.

Understandings of the particular hormones and neurotransmitters associated with autistic spectrum disorders enable consideration to potential deficits in social information processing, and the related possible impact on social interactions in particular contexts or situations. Young people with autism have been found to have lower levels of the neurotransmitter serotonin than those without autism (Chugani, et al, 1999), whilst Spratt et al (2012, p.75) identify an 'increased reactivity of the HPA [hypothalamic-pituitary-adrenal] axis to stress and novel stimuli in children with autism.' Both of these biological components are associated with an increased likelihood of inappropriate aggressive behaviour in novel or stressful situations. Van Goozen, Fairchild, Snoek and Harold (2007, p.162) highlight the association between serotonin and aggression, with low levels of this neurotransmitter 'thought to lead to behavioural disinhibition and distractibility'. In turn, serotonin interacts with the HPA axis, which is implicated in stress response mechanisms, and therefore pertinent to behaviour such as reactive aggression, and is key to the appropriate assessment of emotional social cues (Crockett, 2009). Thus certain forms of aggressive or antisocial behaviour amongst young people with autism may be considered as a product of specific insult-related abnormalities impacting upon cognitive-executive and social-affective functions, resulting in aggressive social behaviours in particular types of social interactions.

This example also highlights the importance of social context in understanding the expression of particular neurodevelopmental disorders and their potential impact on offending behaviour. Specific neurodevelopmental disorders are not expressed in a clear, consistent manner. Instead their expression is seen to be dependent on social interaction and adjustment to social environment, and therefore dependent on context. In a developmental psychopathology framework this is accounted for through consideration to the behavioural tendencies that might be expressed in particular social interactions, and the influence of the

perception of others on an individual's social adjustment. Criminological models that assume a consistent or stable behavioural tendency are therefore unable to adequately explain the particular impact of specific neurodevelopmental difficulties in certain social contexts. Instead, antisocial behaviour must be seen as the result of social interactions, and not just a product of intrinsic factors regarding components of social information processing. Once again, this demonstrates the ready integration of developmental psychopathology with the premises of biosocial criminology in recognising the potential for a biological disadvantage to increase risk of inappropriate behaviour in specific criminogenic contexts.

In criminological research such contexts are clearly those in which offending may occur, such as unsupervised peer group interactions (Farrington, 2002; Osgood and Anderson, 2002; Osgood, Wilson, Bachman, O'Malley, & Johnston, 1996). Various studies indicate challenges in peer group formation, and associated susceptibility to bullying, negative peer pressure and delinquency amongst those with neurodevelopmental disorders. Baldry, Dowse and Clarence (2011) suggest that those with cognitive impairments may have a 'tendency… to want to be accepted by their peer group', and to therefore engage in criminality if associating with criminal peers. This is apparent in relation to young people with a developmental language disorder, with poor use of language and associated limited social skills making the formation of relationships with peers difficult, reducing the capacity for peer negotiation and effective interaction (Botting & Conti-Ramsden, 2000), resulting in young people with speech and language difficulties to be around three times more likely to report 'being regular targets for victimization' when compared to those without such developmental difficulties (Conti-Ramsden & Botting, 2004). Similarly, in a sample of 300 young people with ADHD, Mrug et al (2012) demonstrates that peer rejection predicts subsequent delinquency, while Gudjonsson et al (2008) argue that young people with ADHD

are ‘more compliant in their temperament’ and may therefore be more susceptible to peer influence, whether negative or positive. These findings are further replicated with students with intellectual disability (Baumeister, Storch & Geffken, 2008, Mishna, 2003).

As well as variation in expression in different contexts and situations, in considering the ‘developmental trajectories that occur within these domains’ (Yeates et al, 2007, p.536), models of developmental psychopathology also make it clear that neuropsychological characteristics are not static or fixed, but vary over time. General frameworks, such as that of Yeates and colleagues (2007), are paralleled by models of developmental psychopathology related to the aetiology and progression of specific neurodevelopmental disorders[2]. For example, as presented in Figure 2, Schmidt and Petermann (2009) provide a developmental model regarding ADHD which maps the typical persistence and desistance of certain behavioural traits over time, and outlines the biological and social determinants of the specific progression of the disorder. This supports an understanding of the biological expression and progression of the disorder across the life course, including at typically key points in offending trajectories such as in childhood (where a proportion of LCP offenders are known to begin demonstrating problematic behaviour), in adolescence (when offending typically peaks) and in the transition into adulthood (when offending typically desists for the majority, and where LCP offenders may again be apparent). In relation to ADHD, this includes the emergence of key symptoms of inattentiveness, hyperactivity and impulsivity in early childhood, the particularly pernicious potential impact of substance use in adolescence,

[2] For a comprehensive coverage of the literature, see the ‘Handbook of Developmental Psychopathology’, an edited collection by Lewis and Rudolph (2014), which includes chapters on a range of specific neurodevelopmental and other psychiatric disorders.

and the emergence of further specific symptoms in adulthood, including ‘affect instability’ and ‘extreme emotional reactions’ (Schmidt & Petermann, 2009).

INSERT FIGURE 2 AROUND HERE

In considering the progression of the disorder, Schmidt and Petermann's (2009, p.59) model is explicitly concerned with points in the ‘developmental pathway’ at which particular ‘qualitative changes’ in the expression of the disorder can be observed, and with examining the ‘reasons for these qualitative changes’. This includes the significant effect of initially entering school and the particular challenges of transition into adulthood. At such points a changing social context can be seen to alter the expression of the disorder, whilst the existence of the disorder has a parallel impact on the experience of that social context. This supports analysis of how young people with neurocognitive or neuropsychological impairments cope with these transitions and new contexts, which is influenced by the particular developmental course of a disorder. For example, a review of the literature by Daley and Birchwood (2010) illustrate the common impact of ADHD on early educational experiences. A variety of symptoms associated with ADHD are seen to potentially inhibit ‘school readiness’: ‘impulse control, attentional capacity and hyperactivity’ are seen to ‘hinder [the] ability to acquire crucial skills such as focusing on teachers, interacting with peers and authority figures, and learning emergent literacy, mathematics and language’, while associated executive functioning deficits are found to cause ‘problems with memory, reasoning, academic skills, conceptual development, general cognitive ability’. A subsequent association between ADHD and poor behaviour in school is also well established. Studies comparing samples of children with ADHD to control groups suggest a heightened risk of school suspension or expulsion at each stage of their educational career (Bauermeister et al,

2007, LeFever et al, 2002). This demonstrates the potential cumulative impact of ADHD on educational engagement and the potential for a neurodevelopmental disorder to indirectly influence the risk of criminality by increasing exposure to other known risk factors for offending. In doing so, it demonstrates the potential of symptoms of neurodevelopmental impairment to act as biological risk factors that increase the likelihood of exposure to environmental risk factors for antisocial behaviour, and therefore illustrates the integrative nature of developmental psychopathology and biosocial criminology.

Adopting a developmental psychopathology framework also supports an understanding of the heterogeneity of experience of any specific clinical disorder. By considering symptoms as a series of 'insult related' risk factors, and subsequently modelling the impact of specific factors on components of social competence, the variety and complexity of expression of particular disorders can be considered. This also supports a shift away from isolated clinically defined disorders to a consideration of symptoms or dimensions of disorders, in keeping with the approach of DSM-V. Similarly a developmental psychopathology framework supports consideration to the interaction or overlap between multiple disorders. For example, the developmental model of ADHD advanced by Schmidt and Pettermann (2009) maps prevalent comorbid behavioural disorders (such as oppositional defiant disorder and conduct disorder), affective disorders and personality disorders. This includes the identification of specific time periods of vulnerability, and their parallel progression across the life course.

Developmental psychopathology can further support biosocial models of criminal behaviour amongst young people with neurodevelopmental disorders by supporting the selection of more appropriate variables, including in relation to biological, environmental and 'secondary' risk factors. Understandings of the particular biological components of specific

neurodevelopmental disorders and their subsequent impact on component aspects of social functioning continue to emerge and inform models of developmental psychopathology that can in turn inform the selection of more specific biological variables within biosocial models of criminal behaviour. For example, continuing improvements to brain imaging techniques provide greater insight into the role of specific regions of the brain in relation to particular executive and cognitive functions, including the particular aspects of the brain affected by particular disorders (see, for example, Cherkasova & Hechtman, 2009 and Vogan et al, 2014), and the mapping of complex interactions between different regions of the brain (Anderson & Beauchamp, 2012).

In keeping with models of biosocial criminology, developmental psychopathology supports consideration to the role of social and environmental factors in determining the progression of a neurodevelopmental disorder, and in particular their interaction with 'insult related' biological factors. This in turn supports a modelling of how social and environmental factors interact with biological factors to influence specific components of social functioning. For example, a study tracking the ten year developmental trajectories of young people who experienced TBI during their school years highlights specific social and environmental factors that correlate to its long term impact (Anderson, Godfrey, Rosenfeld, & Catroppa, 2012). The study found that long-term behavioural outcomes were predicted by 'family function' and 'family intimacy' (Anderson et al, 2012, p.259); a result 'in keeping with previous reports from school-age samples and evaluation more proximal to time of injury.' (Anderson et al, 2012, p.259, citing Catroppa et al, 2008 and Yeates et al, 2010). Specifically approaches to parenting that are excessively permissive or authoritarian may be adopted in response to the challenges of parenting a child with challenging behaviour resulting from childhood TBI.

This example illustrates the potential for an initial cognitive dysfunction to result in subsequent social or environmental responses that independently increase the risk of engagement in offending, and therefore the potential indirect influence of neurodevelopmental impairment on criminality. This twofold relationship was also apparent in the above discussions of the influence of neurodevelopmental impairment on educational and peer group experiences know to be risk factors for future offending. Indeed, Anderson et al (2012, p.259) found that 'injury factors, such as severity and brain pathology… become less important with time since injury', with social and environmental factors more influential. This suggests that, in some instances, the direct influence of biological factors related to neurodevelopmental impairment on offending behaviour may be less significant than their indirect influence via other prior social outcomes and experiences. However to date such studies have not been typically designed so as to effectively account for the dynamic influence of this variety of factors on offending careers. Only by combining developmental psychopathology with biosocial criminology can the multifarious and complex influences of neurodevelopmental impairment on trajectories into serious and persistent offending be realized.

Conclusion

Given the established high prevalence of a range of neurodevelopmental disorders amongst young people in custodial institutions in various nation states, it is crucial that criminological research seeks improved understandings of the influence of neurocognitive and neuropsychological impairments on trajectories of offending. Biosocial criminology is clearly aware of such influences, as is evident in the conceptualization and explanation of life course

persistent offending. However, this article has argued that current approaches within biosocial criminology have yet to adequately model or explain the complex relationships between childhood neurodevelopmental difficulties and trajectories of offending behaviour, simplifying complex disorders into variables assumed to be static, consistent or homogenous.

By utilising readily compatible models and understandings from developmental psychopathology, it is possible to both address the observed limitations in existing biosocial criminological research and to identify further particular strands of research enquiry that are not currently readily considered in biosocial criminological models. In particular it supports the selection of appropriate variables, including biological, social and environmental factors known to be key to the etiology, progression and expression of particular disorders. Developmental psychopathology therefore offers a means to integrate understandings of the expression and progression of neurodevelopmental impairment with the principles and foci of biosocial criminology. Specifically it supports an understanding of how symptoms related to neurodevelopmental impairment may act as biological risk factors to increase the likelihood of violent or antisocial behaviour in a specific criminogenic context. Likewise it provides insight into how these biological risks may increase exposure to environmental risk factors for antisocial behaviour. This includes consideration to the impact of impairment on secondary risk factors for offending, including family functioning, educational engagement and peer group interaction, and a necessary 'qualitative gaze' at key points in the developmental course at which neurodevelopmental disorders may find particular expression or acceleration.

Modelling these factors within a heuristic framework, such as that provided by Yeates et al (2007), supports consideration to explanatory pathways or models that can theorise the

influence of specific factors on emergent behaviour in particular social contexts, including those that are criminogenic or associated with specific types of offending. Once modelled effectively these understandings have a clear potential to influence criminal justice practices regarding early and preventative intervention, assessment of underlying needs relevant to offending behaviour, and youth justice practices that are responsive to these needs (Hughes et al, 2012).

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Figure 1. The 'integrative, heuristic model of social competence' developed by Yeates et al (2007)

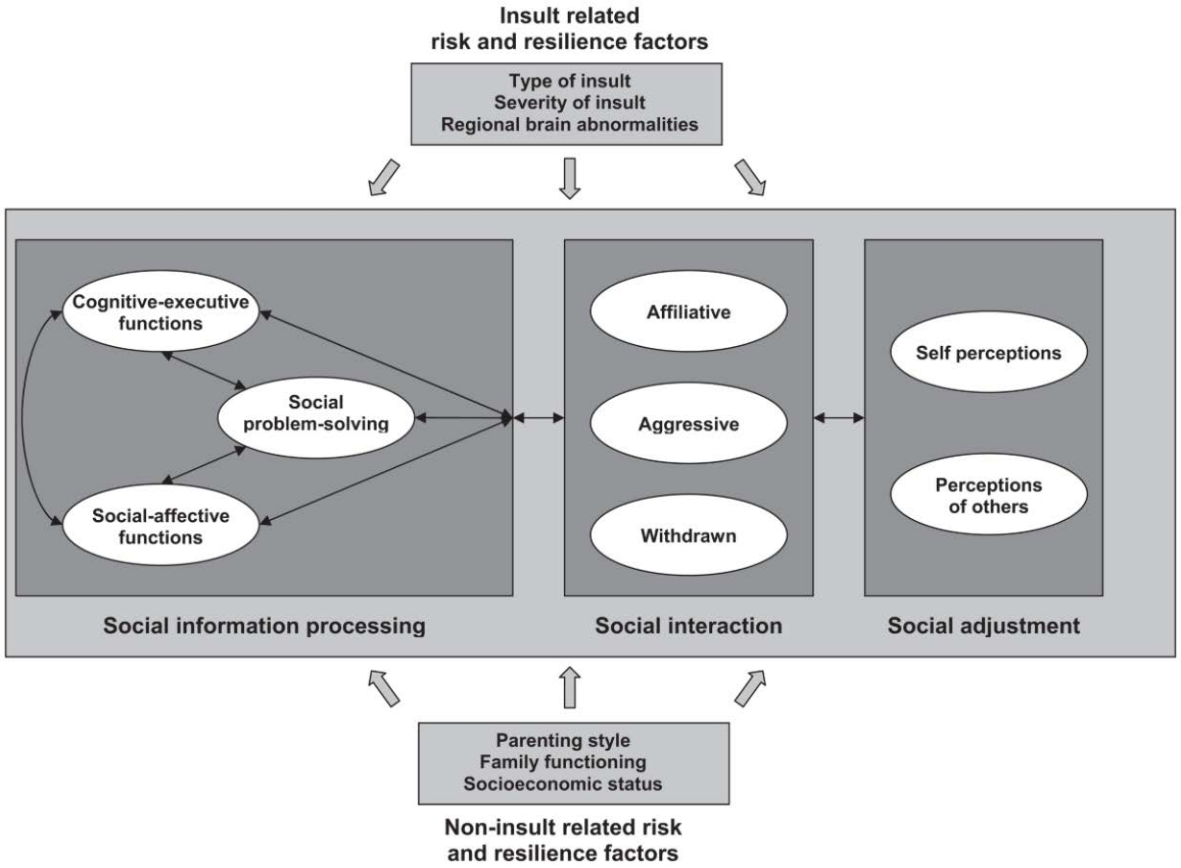

Table 1. Prevalence of neurodevelopmental disorders (adapted from Hughes et al, 2012)

Neurodevelopmental disorder	Definition (based on APA, 2013)	Prevalence rates amongst young people in	
		the general population	custody
Intellectual Disability (also commonly referred to as Learning Disability)	Impairments of 'general mental abilities' that impact adaptive functioning in relation to everyday tasks in one or more of three domains: - the 'conceptual' (including 'language', 'reading', 'writing', 'mathematics', 'reasoning', 'knowledge', and 'memory'); - the 'social' (including 'empathy', 'social judgment', 'interpersonal communication skills', and 'the ability to make and retain friendships'); - the 'practical' (including 'personal care', employment skills, financial management, and 'school and work tasks')	2 - 4%	23 - 32%
Communication Disorders	Problems with speech, language or hearing that significantly impact upon an individual's academic achievement or day-to-day social interactions. corporates a range specific disorders related to: - language (expressive and receptive-expressive) - speech sound / phonology - fluency / stuttering - social (pragmatic) communication, i.e. difficulties in the use of verbal and nonverbal communication in social interaction	5 - 7%	60 - 90%
Attention Deficit Hyperactive Disorder	Persistence in symptoms of: - inattention (such as: failure to give close attention to details; difficulty sustaining attention in tasks; apparent failure to listen when spoken to directly;	1.7 – 9%	12%

	failure to follow through on instructions; difficulty organizing tasks or completing task required sustained effort; easily distracted); and - hyperactivity and impulsivity (such as: fidgeting; restlessness; an inability to play quietly; talking excessively; interrupting others)		
Autistic Spectrum Disorder	'Persistent deficits in social communication and social interaction', such as in: 'social-emotional reciprocity'; 'nonverbal communicative behaviors' and 'developing, maintaining, and understanding relationships' 'Restricted, repetitive patterns of behavior, interests, or activities', such as: 'stereotyped or repetitive motor movements, use of objects, or speech; 'insistence on sameness, inflexible adherence to routines, or ritualized patterns'; 'highly restricted, fixated interests'; 'hyper- or hyporeactivity to sensory input'	0.6 – 1.2%	15%
Neurodevelopmental Disorder - Prenatal Alcohol Exposure (commonly referred to as Foetal Alcohol Syndrome)	Permanent birth defects resulting from prenatal alcohol exposure. Traits include: - physical characteristics (such as facial features; reduced height, weight, and/or head circumference) - impaired neurocognitive functioning (such as behavioural inhibition; poor planning; poor visual-spatial reasoning) - impaired self-regulation (such as mood regulation; attention deficit; impulse control) - impaired adaptive functioning (such as delayed acquisition of language; problematic social communication)	0.1 – 5%	10.9 - 11.7%

Traumatic Brain Injury[3]	Any injury to the brain caused by impact. Depending on the severity and site of the impact, TBI can lead to permanent or temporary impairment of: - cognitive functions (such as attention and concentration; communication; controlling impulses; problem-solving) - physical functions (such as coordination; hearing; seizures; dizziness; headaches) - psychosocial functions (such as depression; anxiety; decreased social contact)	24 - 31.6%	65.1 - 72.1%

[3] Traumatic brain injury is not defined as a psychiatric disorder within DSM-V, but is included here due its similarity in expression to defined disorders, and its prevalence in offending populations